CASE REPORT

A CASE REPORT AND OVERVIEW OF CARBAMATE INSECTICIDE (BAYGON®) POISONING

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ABSTRACT

The following is a case report of a 55-year-old gentleman who experienced excessive exposure to Baygon®, a local carbamate insecticide spray. He developed symptoms that were compatible with cholinesterase inhibition, namely headache, light-headedness, confusion, bad taste, nausea and fatigue. This report demonstrates that the misapplication of insecticides commonly used in residences can cause acute poisoning.

Keywords: insecticides, carbamate poisoning, Baygon®

INTRODUCTION

Poisoning refers to the damaging physiological effects of inhalation, ingestion, skin contact or other exposure to pharmaceuticals, drugs and chemicals, including pesticides, heavy metals, gases/vapours and common household cleaning substances.[1] Poisoning is a real health problem in every country of the world.

Carbamate pesticides are derived from carbamic acid and kill insects in a similar fashion as organophosphate insecticides. They are widely used in homes, gardens and agriculture. Their mode of action is by inhibition of cholinesterase enzymes, affecting nerve impulse transmission. The first carbamate, carbaryl, was introduced in 1956 and has been most widely used throughout the world, more than all other carbamates combined.[2] Most of the carbamates are extremely toxic to foraging parasitic wasps, ants and bees.

CASE REPORT

A 55-year-old gentleman was admitted to the emergency department of Gozo General hospital suffering from sudden onset of headache accompanied by very bad taste, severe dizziness, bouts of confusion and light-headedness. He had no difficulty with breathing. He did not cough or vomit. His past medical history was unremarkable with no history of head injury, nose trauma, diabetes or asthma. According to his wife the episode started 45 minutes before admission while he was helping her prepare dinner. On further questioning he admitted that he had sprayed half a bottle of baygon insecticide in the cellar to which he remained exposed for 20 minutes.

On examination, the patient, who was a well-built man, was conscious but not oriented. Oxygen saturation was 98%. His pulse was regular at 78 beats/min and his blood pressure was 139/88 mmHg. Neurological examination was unremarkable. Laboratory results showed a leucocytosis of 15.3x10(9)/L (range from 4.3 to 9.8x10(3)/L). Liver function and kidney function tests were normal. Arterial blood gas analysis showed metabolic acidosis with pH 7.31, pCO2 of 31 mmHg, pO2 of 96 mmHg and bicarbonate of 16 mmol/L. An urgent CT scan of the brain and paranasal sinuses was done immediately on admission and was reported as normal. The chest X-ray showed no abnormalities. An electrocardiogram showed no conduction abnormalities. A preliminary diagnosis of poisoning due to inhalation of Baygon® insecticide was made. The patient was admitted to the intensive therapy unit. Continuous cardiac monitoring as well as continuous oxygen support and chest physiotherapy were done. Fortunately the clinical signs and symptoms did not require anticholinergic treatment. After two days at the intensive care unit the patient was stable and was transfered to the general ward and eventually discharged home after four days.

DISCUSSION

MECHANISM OF TOXICITY

The signs and symptoms of carbamate poisoning are similar to those caused by organophosphate pesticides. The carbamate's principal route of entry is by inhalation or ingestion or secondarily through dermal route. Dermal exposure tends to be less toxic than inhalation or ingestion.[2] The carbamates are hydrolysed enzymatically by the liver and the metabolites are excreted by the kidneys and the liver. As with organophosphates, the signs and symptoms are based on excessive cholinergic stimulation. Unlike organophosphate poisoning, carbamate poisoning tends to be of shorter duration. This is because the inhibition of nervous tissue acetylcholinesterase (AChE) is more easily reversible. The carbamylation of the enzyme is unstable, and the regeneration of AChE is relatively rapid compared with that from a phosphorylated enzyme. Because of this, carbamate pesticides are less dangerous with regard to human exposure than organophosphorus pesticides. In keeping with this, the ratio between the dose required to produce death and the dose required to produce minimum symptoms of poisoning is substantially larger for carbamate compounds than for organophosphorus compounds.[2]

CLINICAL PRESENTATION

The most commonly reported early symptoms are muscle weakness, dizziness, sweating and slight body discomfort. Higher levels of exposure present with headache, salivation, nausea, vomiting, abdominal pain and diarrhoea. Contraction of pupils with blurred vision, incoordination, confusion, muscle twitching and slurred

speech have also been reported. Respiratory depression together with pulmonary oedema is the usual cause of death from poisoning by carbamate compounds. Because of their chemical structure, carbamates do not cause delayed neuropathy.[3]

FIRST AID MEASURES

***First aid for the eyes*:** The eyes should be rinsed gently with water, ideally running tap water for 15-20 minutes. Contact lenses should be removed if present.

First aid for the skin: Contaminated clothing should be removed and the skin rinsed immediately with plenty of water for 15-20 minutes. The patient's clothes should be discarded since they absorb carbamate agents, and re-exposure may occur even after washing.

***First aid for inhalation*:** The person should be moved to an uncontaminated area so that the patient can breath fresh air. If the person is not breathing one should call for an ambulance then start artificial respiration, preferably mouth–to-mouth, if possible.

TREATMENT OF ACUTE TOXICITY

In our case the patient only needed continuous 100% oxygen via facemask and cardiac support at the intensive therapy unit with spontaneous recovery. In moderate to severe cases the following treatment may be needed.

ATROPINE

Atropine should be given beginning with 2mg IV repeated at 15 to 30-minute intervals. Once atropinized, a maintenance dose at 1-3mg half hourly is usually sufficient. The dose and the frequency of atropine treatment varies from case to case, but should maintain the patient fully atropinized (dilated pupils, dry mouth, skin flushing, normal pulse rate and good mental state). The total dose and duration of atropine use depends on the type and amount of carbamate compound consumed.[4]

VENTILATORY SUPPORT

Intubation is strongly considered in moderate to severe poisoning. Patients who appear mildly poisoned may rapidly develop respiratory failure due to a combination of CNS depression, niconitic receptor-mediated diaphragmatic weakness, bronchospasm and copious secretions.[4]

OXIME REACTIVATORS

There is no rational basis for using these drugs. Furthermore, some unconfirmed reports suggest an increased toxicity of carbamates when oximes are administered.[4]

BENZODIAZEPINE THERAPY

Diazepam 0.1-0.2 mg/kg IV can be given and repeated as necessary if seizures occur. The early use of diazepam may reduce morbidity and mortality.

FLUID AND ELECTROLYTE BALANCE

Patients may require extra fluids and electrolytes to compensate for the loss due to vomiting, high fever, diarrhoea and for decreased intake. Other diseases like diabetes, hypertension, heart failure or complications like aspiration pneumonia have to be dealt with and treated.[5]

SYMPATHETIC AND CARING DISCUSSION WITH PATIENTS

Self-poisoning using carbamates is very common in developing countries. Since first described as a problem in India over 40 years ago, this problem has increased with India and Sri Lanka being the countries with the highest number of cases. Death from pesticide poisoning was the 6th most common cause of death in Sri Lanka in 2003.[4] Patients who survive self-poisoning require an empathic and caring approach by the treating doctors and nurses. These patients may require referral for psychiatric treatment.[5]

GASTRIC LAVAGE AND ACTIVATED CHARCOAL

Emptying the stomach by gastric lavage is most useful if attempted within 1 to 2 hours after ingestion of the poison. If the patient is unconscious, the time elapsed since ingestion may be less relevant since the gastrointestinal stasis which often accompanies coma can delay gastric emptying. It is therefore recommended that gastric lavage is carried out in every unconscious poisoned patient.[6]

CONCLUSION

Pesticides are widely used in buildings and agriculture throughout the Maltese islands. This case report is intended to draw the attention of clinicians to the potential exposure of carbamates and organophosphate pesticides which may cause poisoning.

Manufacturers need to be persuaded to compulsary market their pesticides in tamper-proof packages along with labels mentioning their poisonous nature, contents and antidote in the unfortunate instances of accidental exposure, in conformity with international standards. Legislation and law enforcement against substandard packaging is warranted.[7]

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